

by the committee of the fund, consisting of no less than eight and no more than eighteen directors, one-half by employers, the other half by the employees.

25. A *Reserve Fund* is to be accumulated by the board of directors; 1-20 of the annual income until reserve equals 1-6 of the total expenditure for the preceding three years.

26. The employer must *pay* to local or trade fund, at least monthly *contributions* due from him and his employees, deducting the worker's dues from his wages.

27. *Membership in Fund.* Every person subject to insurance must be a member, unless he be a member of an approved society.

28. An *Approved Society* may be a labor union, benevolent or fraternal society, or an establishment society, if approved by the commissioner.

Such society must not be carried on for profit; must not be under control of insured members; must be in a sound financial condition; must not endanger the existence of any local or trade fund.

29. *Employer's Contributions* shall be the same to the fund, though employees are insured in labor unions, benevolent or fraternal societies.

30. The *State* is to *contribute* to every approved society 1-10 of the total expenses.

31. A *Health Insurance Union* may be formed from two or more insurance carriers controlled by the Commission.

32. The *Guarantee Fund* consists of 10% reserved by the Commission from the contribution of the state to the carriers for the relief of any carrier if an emergency should arise in the judgment of the Commission, because of epidemics or other unusual conditions.

33. *State Supervision.* The *Social Insurance Commission* is to consist of three Commissioners appointed by the Governor, one of whom is to be a physician. Each Commissioner is to devote his entire time to the duties of his office.

34. The Commission shall appoint a *Secretary*, officers, and other assistants, and employees as may be necessary. All of them to be under Civil Service rule.

35. The Commission is to receive a *salary*; the Commissioners and the subordinates are entitled to actual and necessary expenses while traveling on business, the expenses are to be paid out of the State Treasury upon vouchers signed by at least two Commissioners. The office shall be in the capitol, and branch offices as advisable in other cities.

36. Any *Commissioner* can conduct inquiry or hearing, etc., which the Commission is authorized to do, if approved by the Commission.

37. The Commission is to make a *report* to the Governor which he shall lay before the Legislature, giving statistics of their work, and recommendations if desired.

38. The *State Social Insurance Council* shall consist of twelve members; an equal number from employers and employees.

39. The *State Council* is to elect its own president; the Secretary of the Commission is to act as secretary of the Council.

40. The annual report of the Commission and its recommendations shall be laid before the Council, before transmission to the Governor. The Council may approve them or make a separate report.

41. A *State Medical Advisory Board* shall be chosen by the state medical societies. It is to be consulted on medical matters.

42. *Disputes shall be settled* by the Social Insurance Commission on appeal from proper authority or carrier. The Commission may assign any dispute to a *Dispute Committee*, composed of employer and employee members of the Council and a member of the Commission as chairman. Either party may appeal from Dispute Committee to the Commission within 30 days.

43. *Medical Disputes* regarding benefits appealed to Commission shall be referred to the Medical Advisory Board, which is to report to the Commission.

44. *Suits at Law* which can be appealed to the Commission shall not be taken to court until decided by the Commission.

## Original Articles

### TUBERCULOSIS OF THE EYE.

By PHILIP H. PIERSON, M. D., San Francisco, Cal.

The object of this paper, written as it is by an internist, is four-fold—(1) to briefly touch on the blood supply and lymphatics of the eye, illustrating the probable method of infection and spread of tuberculosis; (2) to describe the main characteristics of the most frequently encountered tubercular lesions; (3) and of chief importance, the method at our disposal for diagnosing stubborn and puzzling eye conditions where tuberculosis should be considered; and, lastly, treatment and prognosis.

Tuberculosis in the eye as in other parts of the body, is spread in one or all of three ways; chiefly by the lymphatic system producing tubercles with their attendant consequences—enlargement, caseation and breaking down, less frequently by the blood stream transmitting the toxins more often than the bacilli to vascular organs (except in military tuberculosis), and, lastly, by contact. This last method is more correctly the local progress of the disease than its entrance into one or more parts of the body.

First let me take up the lymphatics of the eye. In the lids there are lymphatic channels anterior and posterior to the tarsus which intercommunicate and drain to the preauricular, submaxillary and anterior cervical glands. It is for this reason that these glands are generally involved, in lesions of the lids proper or the conjunctivae. The cornea contains practically no blood vessels or lymphatics, but there are clefts between the layers of the epithelium which are easily invaded by toxins or bacteria circulating in the blood vessels which are present and terminate in anastomoses at the corneal edge of the sclera. It is probably in this way that lesions occur in the cornea. They may also be the result of contact with the iris when this is involved. Another important lymph circulation is found in

the blood vessel walls of the choroid. These verticose veins have an endothelial lining but no muscular coat whatsoever. Outside this endothelial lining there are lymph spaces—the so-called perivascular lymph spaces of His. Is it not very probably due to these lymphatics rather than the blood stream that we find tubercles singly or miliary of the choroid and from this diseased condition of the choroid the so-called retinitis proliferans with hemorrhage in youth. These perivascular lymph spaces communicate and make their way about the lamina cribrosa into clefts in the sheath of the optic nerve. From this point they penetrate into the lymph spaces existing between the pia, arachnoid and dura, which are continuous back over the brain surface. There are channels in the sclera which intercommunicate and pass to the outer surface of the sclera and drain extradurally. It is by this path that tubercles of the choroid occur in the terminal stages of tubercular meningitis. Whether or not the hyaloid canal exists after fetal life as a communicating canal from the anterior to the posterior part of the eye is a question. Iris and ciliary body lesions may be toxic due to the vascular condition of these structures or here we may have a blood born infection. In tubercular lesions of the iris we sometimes find that it is adherent to the cornea (generally if proper care has not been taken to prevent it), and a tubercular lesion in the cornea results from direct extension. This brief description of the lymphatics I trust may make the source and correlation of some of these eye lesions more evident. Allow me here to emphasize the lymphatic system as a harbinger of latent tuberculosis, and the lymphatic glands of the body should always be investigated as the possible and probable focus for eye tuberculosis, even in the face of an evident lung or bone condition.

The frequency with which tuberculosis affects the eye has been estimated as far back as 1885 by P. H. Mules as 1 in 33,000 eye patients, more recently by Eyre as 1 in 2,700, and in 1913 by Stephenson as 1 in every 1000 cases. From this it is easily seen that while it is not a frequent manifestation of tuberculosis it is becoming more frequently recognized. This I believe is due to our increasing knowledge of how tuberculosis spreads and especially to the more general use of tuberculin as a diagnostic and therapeutic agent.

Let us briefly consider tubercular manifestations in the conjunctiva, cornea, iris and choroid as being probably the most frequent sites in the eye. The first definite case of conjunctival tuberculosis was described by Koester in 1873. It is estimated by Eyre as occurring in 1 out of every 3,000 eye cases and by Lagrange as in 1 out of every 7,500 cases. It is a disease generally met with in persons under 30, more often unilateral, and may be primary according to Ayraud, or secondary to extension from the lacrimal sac or elsewhere in the body. A case was reported by Gourfein in which no other tuberculosis was demonstrable and which later developed brain and pulmonary tuberculosis. When primary the organism gains entrance through some abrasion in the conjunctiva. It is found more often on the tarsal conjunctiva and may be one of

three types—nodular, ulcerating or vegetating. If nodular it presents a red, swollen, hard, gristle-like granule, the size of a millet seed. If there are several they may unite, casefy and ulcerate. The edges of the ulcer are irregular and it is covered with a yellowish grey fibrinous film. When this is situated near the palpebral border cicatrizing deformities may ensue. In the vegetating type there is either a cock's comb vegetation or papillary pseudo trachomatous condition. In all these types there are but few reactionary signs or symptoms. The patient may have the sensation of a foreign body in his eye but no pain; his lid may be slightly swollen and the bulbar conjunctiva echimotic. There is lacrimation and a slight muco purulent secretion. There is practically always glandular involvement, taking in first the preauricular glands, then the submaxillary, and later all the cervical chain. This insidious onset is followed by very slow development. The vegetating type is generally benign, but the ulcerating type is serious and, according to Armaignac, is complicated by laryngeal and pulmonary tuberculosis, according to Gourfein by cerebral and pulmonary manifestations, meningeal in the experience of Manz and Hock, and articular and pleuro-pulmonary in the observation of Kalt. A tubercular condition of the conjunctiva may extend from a nasal lupus which is more frequent in children. About 7-8% of all lacrimal ducts excised are tubercular, according to Jaulin, Rollet, and other observers. In lupus glandular involvement is not present except where there is secondary infection. The differential diagnosis in such a condition as described above, lies between:

Granular conjunctivitis.

Syphilis—a chancre resting on an indurated base and a gumma, which according to Trousseau has no glandular enlargement.

Sporothrix, which can be established by laboratory smears.

Epithelioma.

Parinauds conjunctivitis, which generally heals spontaneously without ulceration, and

Phlyctenular conjunctivitis.

This last condition has and will continue to be the cause of discussion as to whether it is tubercular or not. Pyfer considers it a tuberculous manifestation rather than an actual tubercular lesion. Derby found 73%-83% positive tuberculin reactions in this condition. So far as I have been able to ascertain phlyctenules do not contain tubercle bacilli, nor injected into animals do they cause tuberculosis. They are very frequently found in the scrofulous child, this condition being due to tuberculosis or a chronic focal infection in teeth, tonsils, etc., or in tuberculous adults—more of a reaction to their lack of general resistance than to an actual tubercular disease. The treatment of conjunctival tuberculosis consists in excision of the diseased area when it is local and there are no involved glands, cauterization and sometimes scarification and curetting. I shall speak of tuberculin treatment as a whole in a few minutes.

Interstitial keratitis was described by McKenzie as far back as 1830 as "Scrofulous corneitis." As a primary disease of the cornea it does not exist.

Whether the organisms or their toxins cause corneal lesions is still doubtful, according to Fuchs. Verhoff produced a tuberculosis of the cornea in the eye of a rabbit by injecting dead organisms into the aqueous humor of the anterior chamber and in three months found lesions similar to those seen clinically coexisting with small tubercles of the iris and ciliary body. Clinically it is found together with conjunctival or irido-ciliary lesions. The superficial or ulcerating type is found coexistent with nasal conjunctival lupus or palpebral conjunctivitis. On the cornea there are greyish yellow nodules causing the cornea to look roughened. By their coalescence ulceration takes place. The infiltrated or deep form follows in iris lesions and resembles an interstitial keratitis of luetic origin, differing from it nevertheless in that the latter leaves practically no opacities, while tuberculosis after healing leaves the infiltration of lymphocytes and their resulting haziness. The pathology of these lesions is an infiltration of giant cells surrounded by epithelioid cells and lymphocytes. The differential diagnosis lies between syphilis, leprosy and trachoma.

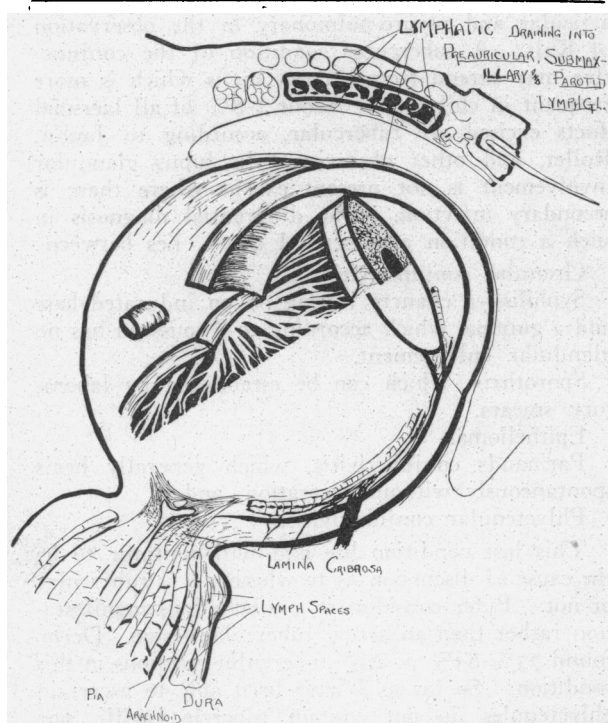


Fig. 1—Anatomy of the Eye with Special Reference to Lymphatics.

I have recently had three cases of tubercular disease of the cornea; two were in patients with advanced pulmonary lesions, and the other in a Hindu with very incipient tuberculosis. The latter had been treated without avail with mercury and Potassium iodide. He presented definitely harsh breathing at his right apex, with a patch of consonating rales in his right axilla. Under careful hygiene and tuberculin his eye quickly cleared up, and remained so until he went back to farming, when he had a recrudescence which again cleared up under tuberculin. One of the other cases was that of a young woman at Arequipa Sanatorium whose eye rather suddenly

became red, the superficial and deep vessels of the conjunctiva and sclera became injected and an infiltration of cells took place. Atropine was used locally, and at first she could not stand the increasing doses of tuberculin because of her extensive lung involvement although she was afebrile. But by repeating the same small dose of tuberculin for a few times she was able to develop more tolerance. Before long the inflammatory reaction had subsided leaving the haziness due to infiltration of lymphocytes and scar tissue. The third case was running considerable fever, and tuberculin could not be used. Her ulceration persisted for a longer time, but finally healed. She had had a similar keratitis when about 18 months old, associated with cervical adenitis, and the old scars in the cornea were visible.

Tuberculosis of the uveal tract is spoken of in the literature as one of the most common forms

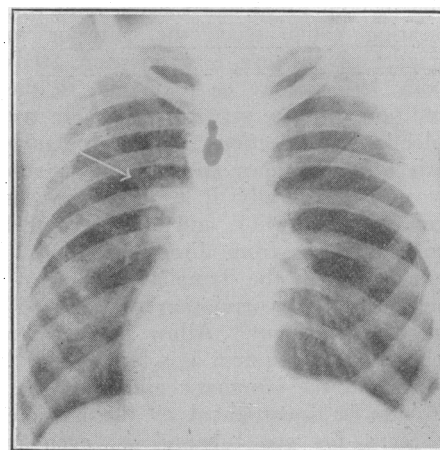


Fig. 11—Bronchial Glands.

of tuberculosis. It may attack the iris, ciliary body and choroid, singly or en masse. Often when the iris is affected the others are involved by continuity, and the cornea and even lens may be involved by pressure. In 1890, Julius Von Michel maintained that inflammations of the iris due to tuberculosis were almost as common as those due to syphilis. This manifestation may be in the form of a solitary or miliary tubercle or it may be a diffuse process due to the toxins and not to the organisms themselves. The condition is practically always endogenous in origin although the individual is frequently in an apparently healthy and even robust condition. 85% of these cases appear before the age of 30, and a large solitary tubercle is more common in the child under ten. This is one of the most serious lesions of the eye, leading often to glaucoma, necessitating removal of the eye for the relief of pain, without influencing the course of the disease from the general constitutional standpoint. The disease in adolescence and adult life is relatively benign but does cause considerable diminution in the acuity of vision from a purely mechanical point of view. In the plastic non-purulent type it is difficult to distinguish from other types of iritis at the beginning, although the inflammatory reaction is less than in syphilis. The progress in

the types other than the solitary tubercle is chronic, tending toward spontaneous healing. Prognosis should be guarded because of the involvement of other tissues by contact, and the dimness of vision resulting. The chief differential diagnosis must be made from syphilis, which when a gumma generally leaves a browner pigment than tuberculosis; lipoma, sarcoma and sporothrix. Few individuals will permit of the excision of a piece for pathological examination and injection into a guinea pig or rabbit's eye. Some of the aqueous humor may be withdrawn and when injected often produces positive results. According to Terrien and Dantrelle, the aqueous humor in tuberculosis is very coagulable. Syphilitic choro-retinitis is more frequently found than is tubercular. The perivascular lymph space of His, it seems to me, plays a very important role as a means of entrance of the disease to the choroid. The diffuseness of the infection gives the appearance of a blood-borne disease, when it is in reality a lymphatic disease. Definite tubercles are found at times in the miliary type when they appear in the ophthalmoscope as fine grayish yellow, round nodules, about one mm. in diameter, with a clear center and a pink border, and may be differentiated from inflammatory nodules by the fact that the latter possess more pigment in their periphery. The hemorrhages that are present in the diffuse process during adolescence are very possibly due to the breaking down of the tubercular disease in the lymphatics, which are in very close proximity to the veins. These generally recover, leaving evidence of the choroidal atrophy. This condition, according to Stock and Axenfeld, is very frequently due to tuberculosis. The occurrence of choroidal tubercles in tubercular meningitis has provoked considerable difference of opinion. Dupuy-Dutemps have never found tubercles in such cases. According to Jackson and others, it is not very uncommon to find these choroidal tubercles in the terminal stages of tubercular meningitis or a miliary tuberculosis, new tubercles appearing every day and of course never attaining any considerable size because of the death of the patient. The lymphatic connection of the choroid by way of the optic sheath and thence to the meninges makes it seem very probable to me that tubercles may be found at such a stage of tuberculous meningitis.

I wish to spend the rest of my time on what seems to me the most important part of this subject, and that is the methods at our disposal for diagnosing chronic, unexplainable eye lesions, and then a few words as to the advisability and usefulness of tuberculin as a therapeutic agent. It seems rather elementary to speak of the importance of a careful history; but this will often throw such light on a doubtful case that other possibilities are considered at once. Of special importance in a possible tuberculous case I would mention the following: (1) general health in childhood, whether always a weak child or strong and robust; (2) glands, these may have subsided with only a small scar remaining; (3) pleurisy; (4) ambition, whether able and anxious to do daily work; (5) the question as to loss of weight. Because of the small focus of possible tuberculosis we can not ex-

pect the more pronounced symptoms of extensive disease. Many times there are points like these that would not occur to the patient himself to mention, but which are of great importance when answered in the affirmative or negative. The general health of most patients with tuberculosis of the eye is remarkably good, and this may be misleading. But the insidious nature of the progress of tuberculosis, the fact that such a small focus is spreading toxins into the system, and the recognition that such a large percentage of us all have at some time been infected, makes it a diagnosis not to be dismissed by the appearance of the individual. A careful physical examination with special reference to general adenopathy, with scars resulting from old discharging wounds in glandular regions, old perforations in the ears which may elicit a story of a chronic discharging ear during childhood; a more rapid heart than can be accounted for by other signs; careful examination of the lungs for an old thickened pleura which may have developed without the frequent pleural pains that are common; suspicious signs of excessive scar tissue in the apex or elsewhere in the lung, and particularly a careful examination of the hilus (bronchial) glands. It is impossible to obtain as much information concerning this most important focus by ordinary physical signs as it is by x-ray pictures of the chest. The plates which I have shown illustrate different hilus shadows and those which I consider of particular importance as of diagnostic value. Whether or not the bronchial glands are the primary source of trouble in the lungs or secondary to some peripheral process, as Gohn claims, I will not discuss here, although I am still inclined toward the former view, they are a very frequent source of trouble and I can not emphasize too strongly a careful examination of them. As to laboratory tests: I believe that as we all use the Wassermann test almost as a routine we should use the subcutaneous tuberculin test in doubtful cases much more than it has been used. Now that I have mentioned tuberculin may I briefly review the different kinds and why one kind may have more value than another. The kinds most frequently used are: Koch's Original Tuberculin, Bacillary Emulsion, Bouillon Filtrate and Tuberculin R. OT is the filtrate of the killed bacilli and hence only the soluble exogenous toxins and none of those insoluble in the bacilli. This stimulates the body cells to form antibodies but has not the bacteriocidal properties that the endotoxins possess. This preparation is concentrated by heat. Denny's Bouillon Filtrate—BF—is similar, except that it has not been heated for concentration. The Bacillary Emulsion—BE—is a preparation of dead organisms and their soluble toxins and so contains exogenous and endogenous toxins. TR is a washed suspension of the dead bacilli and so contains the endogenous toxins. So it can be seen that the bacteriocidal properties are contained only in those with the dead organisms—BE and TR, while BF and OT act more as stimulators to body resisting powers. These are in reality largely theoretical matters and there are good men using almost exclusively one or another of these different preparations as well as many

others, including foreign preparations, and each believes he has better results with that particular kind than with others. OT is used almost exclusively for diagnostic purposes. A great many use TR. At Saranac and elsewhere, equal parts of BE and BF are used in treatment. I favor this last combination because it theoretically at least contains both properties. The question is often raised as to whether it is more efficacious to use Human or Bovine tuberculin. Romer's work on this subject, in which he injected tuberculin made from human, bovine and avian tubercle bacilli into animals infected with human, bovine and avian tuberculosis, and found no difference in the reactions, concluded that "human and bovine tuberculin are so identical in their action on infected animals that we may neglect the source." Koch's original idea as to what caused the febrile reaction to tuberculin was that the injected material had a specific action on the tubercular tissue causing it to slough, and it was the absorption of this necrotic material which caused the rise in temperature. This view is no longer held and now it is considered by Hamman, Wolman and others to be a specific protein anaphylactic reaction involving the protein substance of the bacillus injected or their soluble toxins, and the tubercular tissue. Immunity is produced by the stimulating effect of these endogenous and exogenous toxins on the body to produce more antibodies. We are doing this throughout life with the live organisms that have found their way into our systems, but it has not proven safe or wise to use live organisms in this immunization, for live organisms have been recovered three years after having been injected intravenously into animals. Immunity produced independently of tuberculin or in conjunction with it is only of a relatively amount and duration and an overwhelming infection may easily overcome the existing protection.

I wish to make a plea for the more frequent use of tuberculin in diagnosis and treatment of eye conditions, for it is as specific for tuberculosis as the Wassermann is for syphilis. First, in regard to its use in diagnosis; it may be used on the skin, in the skin or under the skin. The Von Pirquet test is of value, particularly in children and, when it is negative, in adults. Two negative reactions are fairly good proof that there is no active tuberculous disease, although this is by no means final. The most valuable test is the subcutaneous test, and this should not be employed until a thorough physical examination has been made to determine whether there is any latent activity in the lungs or glands. If there is any active disease the diagnostic doses may easily disseminate the process and so do considerable harm. If there is not this danger the doses should be very small at the start, gradually increasing from .1, .3, .7, 1., 1.5, to 2. mgr. A general reaction is not enough, for a great many without eye tuberculosis would react constitutionally to such a dose. But of particular value is the focal reaction in the eye, and to consider the test positive for the eye a change should take place there. After each injection the eye lesion should be examined just as routinely as the

temperature chart. The reaction may take the form of tiny grey deposits or thickening of the iris in the inner circle, according to Professor Stock. Haab has seen ciliary and conjunctival injection in one case of disseminated choroiditis, and hemorrhage near the papilla in another patient from diagnostic doses. In another case where the vitreous had become liquefied and contained numerous dense floculi and the borders of the disc had become very much reddened and there was a white area including the macula, the tuberculin reaction consisted in a hemorrhage in the course of some of the retinal vessels. By this test Axenfeld and Stock found that hemorrhages into the vitreous which commonly accompany retinitis proliferans, were often due to tuberculosis. One of these eyes came to autopsy and while no tubercles were found, the endothelium of the veins was characteristically changed by the tuberculous toxins. Thus a condition often ascribed to pus producing organisms has been shown to be of a tuberculous origin at times. It is much better to use small diagnostic doses in suspicious cases in order to determine the smallest dose which will give a reaction, rather than to do damage by a larger than necessary dose. Gamble has seen a typical reaction in tuberculous iritis with 1/50 mgr., and Stephenson produced a general and focal reaction in two cases of iridocyclitis by 1/1000 mgr. of TR. On the other hand, some cases of definite tuberculosis have not reacted until 6 mgr. were given, but this is far too large a dose to give in safety. A negative focal reaction with a positive general and local reaction is generally sufficient proof that the disease is not tubercular; but I think that there is a great deal of value in the statement of McCool, who says that in spite of a negative focal reaction with a positive general and local reaction where every other possible focus of infection such as teeth, tonsils, accessory sinuses, syphilis, etc., had been excluded, the eye condition should be considered tuberculous and treated as such. Tuberculin and hygiene will build up his resistance to that condition which caused his general reaction, and this will aid in any obscure eye condition.

Finally, in regard to treatment and prognosis: If we consider the eye lesion as probably tubercular we must not put all our faith in tuberculin, without carrying out hygienic measures. This means an inquiry into the habits and occupation of the patient, insisting that his work shall be clean and not fatiguing, that he shall have hours of rest and sleeping out of doors if possible, and that his diet shall consist in plain, simple, nutritious food. This is a very important part of the treatment. Tuberculin is given by one school headed by von Hippel, in doses large enough and frequently enough to produce a slight general and focal reaction. They consider this a necessity for the best immunizing results. This I think is more dangerous than keeping the doses just under the point of giving a reaction. We know that one of the dangers in a reaction is the softening of the tuberculous area and the spreading of the tuberculosis in the blood stream to other parts of the body. Bachmeister examined the blood of fifteen individuals at the

height of the febrile reaction after injection and found the blood positive in four cases. Animal experimentation by Rabinowitch-Kempner found the same to be true. The more conservative school is headed by Wright. He uses BE or TR, starting with 1/80000 or 1/10000 mgr., gradually increasing it at intervals of 10 to 12 days. Von Hippel uses larger doses of BE or TR, beginning with 1/500 mgr., increasing it 1/500 each dose every 2 to 5 days until 1 or 2 mgr. are given. Of course, with either method the patient should be considered individually and no set rule be followed. In using a mixture of BE and BF, I make dilutions up to 1/10,000,000 mgr., gradually increasing the doses till 1/1000 mgr. is reached. This is more applicable to general tuberculosis than to eye tuberculosis, where the dose may be carried further without the dangers of some of the casualties above mentioned. If proper care is taken, these excessive reactions are always avoided. Tuberculous iritis

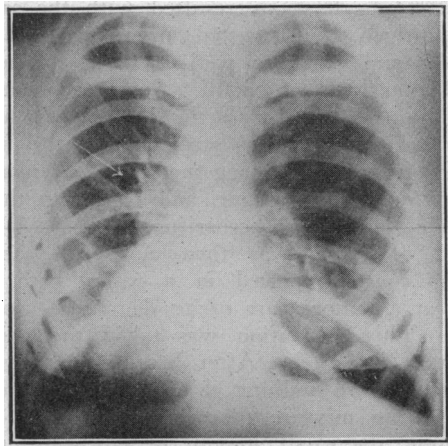


Fig. III—Bronchial Glands.

responds most favorably to this form of treatment and tuberculous conjunctivitis less readily. This may be due to the fact that the conjunctivitis is sometimes a primary source of infection, or it may be due to attenuated organisms. Scleritis, episcleritis and sclerokeratitis also respond favorably to tuberculin. Choroidal lesions respond fairly well, but not as well as do lesions in the anterior segment. One reason for failure sometimes is that it is not continued long enough. At times there is faulty preparation of the tuberculin; another thing it is important to note is that a new set of dilutions is made every 3 or 4 weeks because of its instability. When recurrences take place or relapses occur another kind of tuberculin may be tried with value.

The prognosis should be guarded because of the tendency to recurrence; but the patient can generally be assured that the lesion will not progress further. Many times the clearing up of edema and congestion will give actual improvement in vision. Where there are deformities produced nothing will remove them, and so the acuity of vision is almost always diminished.

In conclusion, let me emphasize the following facts:

(1) The spread of tuberculosis is generally by

means of the lymphatics, while the blood stream transmits the toxins and sometimes the tubercle bacillus.

(2) In considering the diagnosis of tuberculosis, careful attention should be given to the lymphatic system and especially the cervical and bronchial glands as latent foci.

(3) The tuberculin test is of great importance and the reaction in the eye should be watched for after each injection, and the endeavor should be made to use the smallest dose that will produce a reaction.

(4) The treatment should be general as well as with tuberculin. This latter is a very powerful agent for good if used carefully and thoroughly.

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#### COMPLEMENT FIXATION IN TUBERCULOSIS.\*

By BENJ. JABLONS, M. D., and DOVE E. HART, A. B.,  
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To appreciate the factors entering into the Complement Fixation Reaction for Tuberculosis, it is necessary to keep two points in mind; first, the reaction of the human organism to the Tubercle Bacillus and its derivatives, and second, its reaction to the tissue products resulting from the action of the Tubercle Bacillus. It is known that the introduction of a foreign protein of whatever nature into the body calls forth a specific and a non-specific response. The specific reaction is that evidenced by the mobilization of an antibody, whose nature may be that of either an agglutinin, a precipitin, a bacteriotropin, an opsonin, a bacteriolysin or a complement fixing antibody. Then the non-specific antibodies may also be mobilized and these are chiefly of the ferment and anti-ferment variety. In order therefore to diagnose the presence of an organism that is sufficiently active to call forth a response from the infected body, it is necessary to seek for one or even all of these antibodies.

Complement deviation has been found to be the most delicate test for the detection of the presence of an antibody producing substance. Its

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